UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/925,720	08/08/2001	Vincent Giguere	514012000200	4807

25226 7590 10/19/2004

MORRISON & FOERSTER LLP
755 PAGE MILL RD
PALO ALTO, CA 94304-1018

EXAMINER	
WILSON, MICHAEL C	

ART UNIT	PAPER NUMBER
1632	

DATE MAILED: 10/19/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/925,720	GIGUERE ET AL.	
	Examiner	Art Unit	
	Michael C. Wilson	1632	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *16 July 2004*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-19,22-29,32-36 and 39* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *15-19,28,29,35 and 36* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☐ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____.
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Election/Restrictions

This application contains claims 1-14, 22-27, 32-34 and 39 drawn to an invention nonelected with traverse. A complete reply to the final rejection must include cancellation of nonelected claims or other appropriate action (37 CFR 1.144) See MPEP § 821.01. Applicants argue 5,789,654 has claims directed toward transgenics and a method of screening using the transgenic. Therefore, applicants conclude the examiner should recombine Groups I and II in the instant application. Applicants' argument is not persuasive because the burden for each application varies. Each application is evaluated on its own merits. As written, the claims encompass methods of using knockout and knockin mice, each of which have a different structure and phentype.

Claims 20, 21, 30, 31, 37 and 38 have been canceled. Claims 15-19, 28, 29, 35 and 36 remain under consideration in the instant office action.

Sequence Listing

The sequence listing requirements have been met. The sequences on pg 13, line 11, pg 14, line 17, and pg 17, line 3, and in claims 23 and 33 were amended on 4-2-02.

Specification

The first line of the specification has been amended to claim priority to US Provisional Application No: 60/119,024, filed 2-8-99.

Claim Rejections - 35 USC § 101

Claims 15-19, 28, 29, 35 and 36 remain rejected under 35 U.S.C. 101 because the claimed invention is not supported by either a specific or substantial asserted utility or a well-established utility for reason of record.

Claims 15, 16, 18, 19, 28, 29, 35 and 36 are directed toward a method of screening compounds using a transgenic mouse, having a disruption of the endogenous ERRα orphan nuclear receptor, whose genome additionally comprises DNA encoding human ERRα, wherein said mouse has an altered fat metabolism and/or glucose metabolism as compared to a control animal. Claim 17 is drawn to a method of screening compounds using a mouse with a knockout of endogenous ERRα that has altered fat and/or glucose metabolism as compared to a control animal.

Claims 15, 16, 18, 19, 28, 29, 35 and 36 as currently amended are limited to a method of using a knock-in mouse (knockout the endogenous gene while expressing a homologous exogenous gene). Claim 17 encompasses methods of using a knock-out or knock-in mouse.

Chen taught ligands for ERRα were unknown (2001, J. Biological Chem., Vol. 276, No. 30, pg 28465-28470).

The specification teaches making ERRα -/- mice (Example 2, pg 38; Example 4, pg 40, line 11). The ERRα -/- mice had decreased body mass, lower body fat (¶ bridging pg 40-41), a decreased intestinal capacity for fatty acid esterification (pg 43, lines 6-9), normal core temperature, basal metabolic rate and expression levels of

uncoupling protein (pg 44, lines 14-17). The mice were treated with 3H2O and showed decreased lipidogenesis as compared to littermate controls (pg 45, lines 9-10).

Claim 17 has no utility. The specification and the art at the time of filing do not teach how to use the ERRα -/- mice to screen and identify compounds that modulate EERα receptor activity. Compounds that modulate ERRα activity cannot be found using the ERRα -/- mice because the mouse has a disruption of endogenous ERRα. None of the phenotypes of the ERRα -/- mice are specific to a disease or linked to a disruption in the ERRα gene in humans. Therefore, methods of screening compounds using the ERRα -/- mice do not have a specific, credible or substantial use because the mice do not express ERRα and because the methods can be performed in wild-type animals.

Claims 15, 16, 18, 19, 28, 29, 35 and 36 have no utility because the specification does not teach how to use mERRα -/- , hERRα +/+ mice. The specification does not teach making mERRα -/- , hERRα +/+ mice or that mERRα -/- , hERRα +/+ mice have altered fat metabolism and/or glucose metabolism as compared to a control animal as claimed. In fact, mERRα -/- , hERRα +/+ mice may have a wild-type phenotype because the human ERRα is expressed to a normal level and has the same function as the mouse ERRα. mERRα -/- , hERRα +/+ mice may also have a completely different phenotype than mERRα -/- mice because the human ERRα protein may be overexpressed. If mouse and human ERRα have the same function, then the method of using a mERRα -/- , hERRα +/+ mouse with a wild-type phenotype would have no substantial or specific utility over a method of using a wild-type mouse.

Nowhere does the specification teach how to use mice to screen and identify compounds that modulate EERα receptor activity. Compounds that modulate mouse ERRα activity cannot be found using the mouse of claim 8 as claimed because the mouse of claim 8 has a disruption of endogenous ERRα; mouse ERRα activity cannot be assayed in mice that do not express mouse ERRα. Compounds that modulate human ERRα activity cannot be found using the animals of claim 8 as claimed because the activity of human ERRα was unknown; human ERRα activity cannot be assayed in mice without knowing the activity of the protein. None of the phenotypes are specific to a disease or linked to a disruption in the ERRα gene in humans. Applicants do not teach the phenotype of the mice of claim 8 or how to use the mice as controls to identify compounds. Therefore, methods of screening compounds using animals having a disruption of endogenous ERRα and an insertion of human ERRα do not have a specific or substantial use because the methods can be performed in wild-type animals.

Applicants argue the mouse of the invention is used by two companies to "validate the specificity of compounds suspected of acting ERRα. In one particular embodiment, the companies have identified a compound (or pool thereof) suspected of acting on ERRα, use of ERRα knock-out mouse to ensure that the compound in fact acts through ERRα." Applicants argue the mouse is a "powerful control to ensure that a compound suspected of modulating weight gain, lipid metabolism, or other parameters or function of the present invention, is effected via a targeting of ERRα." Applicants' arguments are not persuasive.

First, the claims are directed toward methods of using mice to identify compounds that modulate ERRα; the claims are not directed toward the mouse.

Second, it cannot be determined whether companies use ERRα -/- mice or mERRα -/- , hERRα +/+ mice.

Third, the specification and the art do not teach how to identify compounds that modulate ERRα using the mice. Applicants' arguments do not teach the steps required to identify compounds that modulate ERRα using the mice. The arguments do not include the specific steps used by the companies to identify compounds. The steps required to use ERRα -/- mice or mERRα -/- , hERRα +/+ mice as a "powerful control" in the method claimed (to identify compounds that modulate ERRα) cannot be envisioned.

Forth, identifying compounds that are "suspected of acting on ERRα" using ERRα -/- mice do not correlate to the claims as amended which require using ERRα -/- mice also express human ERRα. Therefore, applicants' arguments do not correlate to the claims.

Fifth, applicants' arguments do not correlate the teachings of the specification with the method utilized by the companies to identify compounds that are "suspected of acting on ERRα".

Finally, compounds that are "suspected of acting on ERRα" may not alter the activity of ERRα as claimed or have any pharmaceutical purpose.

Bowery (Pharm. Rev., 2002, Vol. 54, pg 247-264) taught antagonists of a receptor protein but did not teach the antagonist had any pharmaceutical use. In fact, the antagonist was used in assays to determine the function of the receptor, but no

function of the receptor was found. Therefore, the antagonist did not function as a research tool. Bowery states, "no unique pharmacological or functional properties have been assigned to either subunit or the variants" of $GABA_B$. "The emergence of high-affinity antagonists for $GABA_B$ receptors has enabled a synaptic role to be established. However, than antagonists have generally failed to establish the existence of pharmacologically distinct receptor types within the $GABA_B$ receptor class. The advent of $GABA_{B1}$ knockout mice has also failed to provide support for multiple receptor types" (pg 247, col. 2, lines 4-). Therefore, administering compounds of interest to the mice may not result in indentifying compounds of pharmaceutical value and may not elucidate the role of the protein being knocked out.

Olsen (GABA in the Nervous System, 2000, pg 81-95) taught that "although gene targeting is often useful in delineating the contribution of a given gene product to phenotypic characteristics observed, some gene knockouts lead to embryonic or perinatal lethality, and others lead to no apparent phenotype. This can arise from a lack of any role for the gene in question in regard to the trait studies or from compensation by other gene products. Analysis of the compensation can yield valuable clues to the genetic pathway" (pg 82, last 11 lines of col. 1). Thus, compounds identified using knockout mice may actually act on other proteins compensating for the knocked out protein. If the function of human ERRα (knocked into the mouse as claimed) has a different function than the endogenous ERRα (knocked out of the mouse), the compounds identified may actually be acting act on other proteins compensating for the

knocked out protein. In other words, the method claimed may never identify a compound that modulates ERRα receptor activity as claimed.

Applicants argue the "power of genetics, the knowledge of the genetic makeup of the transgenic animal (such as a mouse), the knowledge of the expression level of ERRα, when used in a screening assay or owtherwise, provides a significant advantage" (pg 15 of response filed 7-16-04). Applicants' argument is not persuasive. The expression level of human ERRα in transgenic mice with a disruption in endogenous ERRα is not described in the specification or in the art at the time of filing. The advantage is not described and cannot be envisioned. The amount of work required to make a transgenic mouse as opposed to a wild-type mouse does not provide any advantage. In fact, wild-type mice provide the advantage of being readily accessible and are more genetically stable than transgenics that can spontaneously excise the transgene.

The metes and bounds of a promoter that is "capable of being modulated by ERRα" in claim 18 cannot be determined. Written description.

Claim Rejections - 35 USC § 112

Claims 15-19, 28, 29, 35 and 36 remain also rejected under 35 U.S.C. 112, first paragraph. Specifically, since the claimed invention is not supported by either a specific or substantial asserted utility or a well established utility for the reasons set forth above,

one skilled in the art clearly would not know how to use mice having a disruption in the ERRα gene as claimed for reasons of record.

The rejection regarding how to make transgenic animals or cells having a disruption in the ERRα gene other than mice has been withdrawn because claim 15, 16, 18, 19, 28, 29, 35 and 36 are dependent on claim 8, which is limited to a mouse and claim 17 is dependent on claim 4, which is limited to a mouse. The term animal in claims 15-19, 28, 29, 35 and 36 should be mouse as in parent claim 4 or 8.

The rejection regarding the correlation between the phenotype obtained in mice to the phenotype obtained in other species has been withdrawn because claim 15, 16, 18, 19, 28, 29, 35 and 36 are dependent on claim 8, which is limited to a mouse with a disruption in endogenous ERRα that expresses human ERRα and claim 17 is dependent on claim 4, which is limited to a mouse with a disruption in endogenous ERRα. The term "animal" in claims 15-19, 28, 29, 35 and 36 should be "mouse".

The specification does not enable using a mERRα -/- , hERRα +/+ mouse with a phenotype of altered fat and/or glucose metabolism in claims 15, 16, 18, 19, 28, 29, 35 and 36 as newly amended. ERRα -/- mice had the phenotype of altered fat and/or glucose metabolism (¶ bridging pg 40-41; pg 43, lines 6-9; pg 44, lines 14-17; pg 45, lines 9-10). However, mERRα -/- , hERRα +/+ mice may have a wild-type phenotype because the human ERRα is expressed to a normal level and has the same function as the mouse ERRα. mERRα -/- , hERRα +/+ mice may also have a completely different phenotype than mERRα -/- mice because the human ERRα protein may be overexpressed. The specification does not teach how much or little of the human ERRα

protein to express to obtain the phenotype of altered fat and/or glucose metabolism in mERRα -/- , hERRα +/+ mice used in the methods of claims 15, 16, 18, 19, 28, 29, 35 and 36.

Claims 15-19, 28, 29, 35 and 36 remain rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention for reasons of record.

It cannot be determined how compounds that modulate ERRα activity can be found using the mice of claim 8 as in claims 15, 18, 28 and 35 because the activity of human ERRα was unknown. The method used to "determine" whether a compound increases or decreases ERRα activity in step b) of claim 15, for example, cannot be determined.

It cannot be determined how compounds that modulate ERRα activity can be found using the mice of claim 4 as in claim 17 because the mice do not have an endogenous ERRα gene. The method used to "determine" whether a compound increases or decreases ERRα activity in step b) of claim 15 cannot be determined.

Applicants argue one of skill would realize numerous means of determing whether an activity of ERRα had been altered because the specification teaches ERRα binds to hormone response elements, interacts with Erα, is involved in numerous physiological processes, interacts with ERRβ and/or ERRγ and affects many physiological and metabolic parameters or functions. Applicants' argument is not persuasive. Binding to hormone response elements is not an activity of ERRα.

"Interacting" with Erα, being "involved" in numerous physiological processes, and "interacting" with ERRβ and/or ERRγ are not activities of ERRα and are generic characteristics that do not set forth how they interact or are involved. The specification does not teach one specific physiological or metabolic activity of ERRα.

The ERRα -/- mice described in the specification have normal body temperature, basal metabolism and uncoupling protein expression; therefore, it is unclear how to use such parameters as in claim 16 to determine whether a compound increases or decreases ERRα activity.

Applicants argue those of skill would know that lipogenesis, for example, correlates to increased or decreased ERRα activity and/or level (pg 45, lines 6-15), while the body temperature could on the other hand not be affected, thereby supporting a specific effect on ERRα." Applicants' arguments are not persuasive. Pg 45, lines 6-15, would not allow one of skill to use body temperature in an ERRα -/- mouse to determine whether a compound altered ERRα activity because the body temperature of the ERRα -/- mouse is normal. The rejection is based on how to use the "normal" ERRα -/- mouse characteristics to determine whether ERRα activity had changed. Applicants' statement "that some parameters (e.g., lipogenesis) indeed will correlate with increase or decrease of ERRα activity and/or level (page 45, lines 6-15), while the body temperature could on the other hand not be affected, thereby supporting a specific effect of ERRα" does not make sense. Lipogenesis may be altered but body temperature is not; however, claim 16 specifically requires determining the activity of ERRα by determining a change in body temperature.

The metes and bounds of "hepatic synthetic functions" in claim 16 remain unclear. The phrase is not defined in the specification and does not have an art recognized meaning. Applicants point to pg 41, lines 1-3, and Table 2, "which define this terminology." Applicants' argument is not persuasive because the phrase is set forth but is not defined, i.e. "mutant animals had normal hepatic synthetic functions."

The metes and bounds of "compounds suspected of being a modulator of ERRα... ...wherein the agent has been identified as a suspected modulator of ERRα activity and/or level in a previous screening assay" in claim 17 remain unclear. It is unclear what level of "suspicion" is required in the previous assay to be considered a candidate for the method claimed, if any compound identified in any previous screening assay is encompassed by the phrase or if the compounds must meet specific criteria before being used in the method. What level must be reached to consider a compound within the metes and bounds of the claim. Where is the line drawn? What specific assays must be used? What functions must the compounds have?

The rejection of claim 18 regarding how compounds that modulate ERRα activity can be found using the mice that do not have an endogenous ERRα gene has been withdrawn because the claim is dependent on claim 8.

The metes and bounds of a promoter that is "capable of being modulated by ERRα" in claim 18 cannot be determined. The metes and bounds of promoters having such capabilities cannot be determined because the specification does not define such promoters or provide an assay for determining such promoters. Nor were such promoters taught in the art at the time of filing.

The "measuring" step of claim 18 is wholly unclear. It cannot be determined what is being measured or selected; i.e. the marker gene or the marker protein, what conditions are required, i.e. the presence or absence of an agent, or how to identify agents that modulate ERRα activity by comparing the marker in the presence or absence of the agent.

The metes and bounds of "agents suspected of modulating the promoter modulating activity of ERRα" in claim 18 cannot be determined. It is unclear if any compound is encompassed by the phrase or if the compounds must meet certain criteria before being used in the method. In addition, the promoter modulating activity of ERRα is unclear as it is not discussed in the specification.

The metes and bounds of "ERRα or related factors" in claim 28 is unclear. How related must the factor be to ERRα to be within the metes and bounds of the claim?

Step a) in claim 28 is unclear. The phrase "which modulates activity thereof by an interaction thereto of said ERRα and related factors" is unclear because it is unclear to what "thereof" and "thereto" refers, i.e. ERRα or related factors vs. the promoter. The structure of the products required in step a) are not clearly set forth.

The phrase "said transcriptional activity" in claim 28, step b) does not have antecedent basis in step a) which only requires "a transcriptionally active preparation of ERRα".

The phrase "or of a binding of at least ERRα or related factors to said cis-acting sequence..." in step b of claim 28 does not make sense. The alternative condition cannot be determined.

The first "measuring" step of claim 28 is wholly unclear. It cannot be determined what is being measured or selected; i.e. the activity of the promoter or of ERRα, what conditions are required, i.e. the presence or absence of an agent, or how to identify agents that modulate ERRα activity by comparing the "activities" in the presence or absence of the agent.

The metes and bounds of "agents suspected of modulating the transcriptional activity of ERRα" in claim 28 cannot be determined. It is unclear if any compound is encompassed by the phrase or if the compounds must meet certain criteria before being used in the method. In addition, the transcriptional activity of ERRα is unclear as it is not discussed in the specification.

The second "measuring" step of claim 28 is wholly unclear. The phrase "and comparing same with that of a control animal, not having been administered" is unclear because it cannot be determined what is the "same." It cannot be determined how to identify agents that modulate ERRα activity by comparing the fat tissue growth or weight gain of an ERRα -/- mouse given the agent with the fat tissue growth or weight gain of an ERRα -/- mouse not given the agent because ERRα -/- mice do not express ERRα. It cannot be determined how to identify agents that modulate ERRα by comparing the fat tissue growth or weight gain of an ERRα -/- mouse given the agent with the fat tissue growth or weight gain of a wild-type mouse given the agent because all wild-type mice are have higher fat tissue growth and weight gain as compared to ERRα -/- mice.

Claim 28 requires using a non-human animal. Dependent claim 30 requires the animal is a mammal. However, dependent claim 31 states the mammal is a human, which is excluded in parent claim 28. Human should be deleted from claim 31.

The metes and bounds of "ERRα or related factors" in claim 35 is unclear. How related must the factor be to ERRα to be within the metes and bounds of the claim?

Step a) in claim 35 is unclear. The phrase "which modulates activity thereof by an interaction thereto of said ERRα and related factors" is unclear because it is unclear to what "thereof" and "thereto" refers, i.e. ERRα or related factors vs. the promoter. The structure of the products required in step a) are not clearly set forth.

The phrase "said transcriptional activity" in claim 35, step b) does not have antecedent basis in step a) which only requires "a transcriptionally active preparation of ERRα".

The phrase "or of a binding of at least ERRα or related factors to said cis-acting sequence..." in step b of claim 35 does not make sense. The alternative condition cannot be determined.

The "measuring" step of claim 35 is wholly unclear. It cannot be determined what is being measured or selected; i.e. the activity of the promoter or of ERRα, what conditions are required, i.e. the presence or absence of an agent, or how to identify agents that modulate ERRα activity by comparing the "activities" in the presence or absence of the agent.

The metes and bounds of "agents suspected of modulating the transcriptional activity of ERRα" in step b of claim 35 cannot be determined. It is unclear if any

compound is encompassed by the phrase or if the compounds must meet certain criteria before being used in the method. In addition, the transcriptional activity of ERRα is unclear as it is not discussed in the specification.

The "assessing" step of claim 35 is wholly unclear. The phrase "and comparing same with that of a control animal, not having been administered" is unclear because it cannot be determined what is the "same." It cannot be determined how to identify agents that modulate ERRα activity by comparing the obesity of an ERRα -/- mouse given the agent with the obesity of an ERRα -/- mouse not given the agent because the mice do not express ERRα. It cannot be determined how to identify agents that modulate ERRα by comparing the obesity of an ERRα -/- mouse given the agent with the obesity of a wild-type mouse given the agent because all wild-type mice are obese as compared to ERRα -/- mice.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the

shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

No claim is allowed.

Inquiry concerning this communication or earlier communications from the examiner should be directed to Michael C. Wilson who can normally be reached at the office on Monday, Tuesday, Thursday and Friday from 9:30 am to 6:00 pm at 571-272-0738.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

Patent applicants with problems or questions regarding electronic images that can be viewed in the Patent Application Information Retrieval system (PAIR) can now contact the USPTO's Patent Electronic Business Center (Patent EBC) for assistance. Representatives are available to answer your questions daily from 6 am to midnight (EST). The toll free number is (866) 217-9197. When calling please have your application serial or patent number, the type of document you are having an image problem with, the number of pages and the specific nature of the problem. The Patent Electronic Business Center will notify applicants of the resolution of the problem within 5-7 business days. Applicants can also check PAIR to confirm that the problem has been corrected. The USPTO's Patent Electronic Business Center is a complete service center supporting all patent business on the Internet. The USPTO's PAIR system provides Internet-based access to patent application status and history information. It also enables applicants to view the scanned images of their own application file folder(s) as well as general patent information available to the public.

For all other customer support, please call the USPTO Call Center (UCC) at 800-786-9199.

If attempts to reach the examiner are unsuccessful, the examiner's supervisor, Amy Nelson, can be reached on 571-272-0804.

The official fax number for this Group is (703) 872-9306.

Michael C. Wilson

MICHAEL WILSON
PRIMARY EXAMINER